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Original Article

BENIGN PAROXYSMAL POSITIONAL VERTIGO IN REHABILITATION SETTING: REVIEW OF DIAGNOSIS AND INTERVENTION

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ABSTRACT

Opinion Statement

Benign Paroxysmal Positional Vertigo (BPPV) is the most common of vestibular disorders, which is usually due to free-floating, misplaced otoliths that have inappropriately entered one of the semicircular canals of the inner ear. It can be diagnosed with great certainty, and in most patients, it can be cured with a simple physical therapy maneuver in which particles simply need to be moved out of the posterior semicircular canal and into a part of the ear where they do not cause symptom.

Keywords: Vestibular Disorders, Misplaced Otoliths, Semicircular Canals

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INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is a common vertigo disorder. Using positional testing, BPPV can readily be diagnosed in the emergency department. There is compelling evidence that free-floating end lymph particles in the posterior semicircular canal underline most cases of benign paroxysmal positional vertigo (BPPV). Recent pathological findings suggest that these particles are orthodontia, probably displaced from monolithic membrane in the utricle. They typically settle in the dependent posterior canal and render it sensitive to gravity.[1-4.]BPPV was first described by Barany in 1921, and he attributed the disorder to otolith disease[5]. Dix and Hallpike described the classic positioning which causes a characteristic nystagmus[6] and it is characterized by brief attacks of vertigo, with associated nystagmus, precipitated by certain changes in head position with respect to gravity[7]. The incidence is difficult to estimate because of the benign, typically self-limited course of the disease. The incidence increases with age and the mean age at onset is in the 4th and 5th decades. Classical symptom of BPPV is the sensation of vertigo usually occurs when you roll or sit up in bed, or even when you bend to pick up an object off the floor. The attack which is classically sudden and violent, last for about half a minute but can leave you feeling “out of balance” for quite some time. The predisposing factors for BPPV include closed head injury,

followed by vestibular neuritis, prolonged bed rest, Meniere's disease, infection and surgical procedures like stapedectomy and insertion of cochlear implant.[8, 9]

BPPV occurs when tiny calcium carbonate crystals (like grains of sand) from one chamber (eolith) where it is perfectly normal, to another semi-circular canal of the inner ear. When the semi-circular canal orientation is changed the crystals roll down to produce brief vertiginous sensations. The mechanism of BPPV is explained by 2 primary theories the first is cupulolithiasis,[10] when dislodged otoconia directly attach to the cupula, and reorientation of the canal relative to gravity deflects the cupula, that exciting or inhibiting the ampullary organ. The second is canalithiasis[11], where otoconia freely sediment in the canalsand reorientation of the canals causes the otoconia to move to the lowest part of the canals, creating a drag on the endolymph, resulting in fluid pressure on the cupula, and activating the ampullary organ.

Symptoms of BPPV

The main symptom is intense dizziness (vertigo) which last for 10-20 seconds and usually no longer than a minute. The vertigo is usually triggered by certain head positions and movements. In addition to vertigo, symptoms of BPPV include imbalance, difficulty, concentrating, and nausea. Activities that bring on symptom can vary in each person, but common head movements include looking up, or rolling over and getting out of bed. It is not considered to be life threatening but it can be tremendously affecting the quality of life of persons due to an increased risk of falls.

Cause of BPPV

The most common cause of BPPV in adult is head injury and is presumably a result of concussive force that displaces the otoconia. In elderly, BPPV is most commonly idiopathic, meaning it occurs for no known reason, and some case it is generally associated with natural age-related degeneration of the otolithic membrane. In middle-aged women, hormonal factors may play a role in the development of BPPV[12]. Due to hormonal disturbance decreased estrogen levels may disturb the internal structure of the otoconia or their interconnections and attachments to the gelatinous matrix and an increase in the concentration of free calcium in the endolymph due to increased calcium resorption may reduce the capacity to dissolve the dislodged otoconia.

In rare condition after mastoid surgery patients develop BPPV. Traumatic BPPV exhibits several distinctive characteristics, look different from idiopathic form including a higher incidence of bilateral, involvement of multiple canals on the same side, more difficult to treat, and frequent recurrences.

In some cases BPPV may develop secondary to any of the inner ear diseases like vestibular neuritis, and Meniere's disease that give rise to degeneration and detachment of the otoconia, but do not totally impaired functions of.[13] BPPV appears to be common (9.8%) in vestibular neuritis patients, and predominantly affects patients who did not fully recover from the disease. BPPV after vestibular neuritis appears to be more difficult to treat than idiopathic BPPV. The incidence of BPPV is also known to be higher in patients who suffer from migraine; even though the exact mechanism remains to be elucidated[14] BPPV has been reported to occur in association with giant-cell arteritis, diabetes, and hyperuricemia[15-17].

Diagnosis and Treatment

Physician can identify on the basis of history, physical examination of affected ear by starting the direction of movement in the form of some vestibular and auditory tests. In the case of orthostatic hypotension dizziness get worse on

standing rather than lying down. Electronystagmography (ENG) testing diagnoses the case characteristic nystagmus (jumping of eyes). Kentala and Pyykko[18] reported that 80% of patients experience a rotatory vertigo and 47% experience a floating sensation. Classically BPPV is diagnosed by observing the patterns of nystagmus induced during positioning maneuvers. But, specific observations of the nystagmus require the fixation to be removed during the maneuvers. As the name implies, BPPV is most often a benign condition and may resolve as time goes on without specific treatment, however, in certain situations it may become dangerous. For example a labor working on the top of building or ladder may suddenly become vertiginous and loose his/her balance, risking a trauma. According to a report of untreated BPPV, most HC-BPPVs resolve within 16 ±19 days and PC-BPPVs within 39 ±47 days of their onset. However, a correct diagnosis and proper rehabilitation repositioning maneuvers may speed up the recovery[19].

POSTERIOR CANAL BPPV (PC-BPPV)

Diagnosis

The diagnosis of posterior canal BPPV is made by performing the **Dix-Hallpike maneuvers** (Figure 1). Diagnostic finding for BPPV of the posterior canal are: Torisonal ocular nystagmus towards the downward ear tested with an upward motion lasting less than 60 seconds, latency between 1 and 40 seconds, and symptoms of vertigo reported by the patients during the Dix-Hallpike maneuver. During this maneuver, the free-floating otolithic debris the posterior canal moves away from the cupula and stimulates the posterior canal by inducing ampullo-fugal flow of the endolymph (Ewald's first law). Excitation of the posterior canal in turn activates the ipsilateral superior oblique and contralateral inferior rectus muscles, which results in tonic downward deviation of the eyes with torsion in the direction of the uppermost ear. Accordingly, the resultant nystagmus would be upbeating and torsional, with the upper pole of the eyes beating toward the lowermost ear.

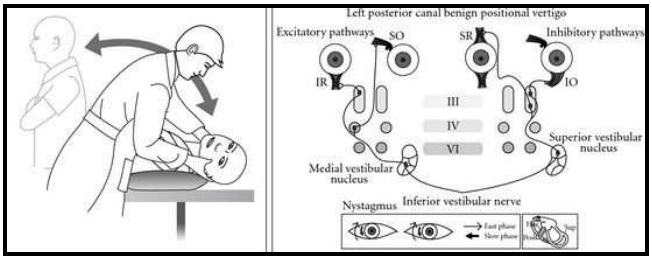

Figure 1: Posterior Canal Bppv in a Left Ear Showing Dix Hallpike Test, Inner Ear, and Receptor Connections to the Extraocular Muscles

The nystagmus is of limited duration, because the endolymph drags ceases when the canalith mass reaches the limit of descent and the cupula return to its neutral position. "Reversal nystagmus" occurs when the patient returns to the upright position; the mass moves in the opposite direction, thus creating a nystagmus in the same plane but the opposite direction. The nystagmus is fatigable with repeated examinations. For the diagnosis of PC-BPPV Dix-Hallpike maneuver has been considered the gold standard. However, we should always perform with caution in patients with a history of neck surgery, cervical radiculopathy, and vascular dissection syndrome, due to its requiring rotation and extension of the neck during the positioning.

Nonsurgical Management

Traditionally, patients were instructed to avoid positions that included their vertigo. Recently the popular methods for treating PC-BPPV are Liberatory Manoeuvre and Particle repositioning Manoeuvre.

Liberatory Manoeuvre This manoeuvre described by Semont and colleagues[20] is indicated for the treatment of PC-BPPV (Figure 2) based on the cupulolithiasis theory. The manoeuvre begins with the patient is seated in the upright

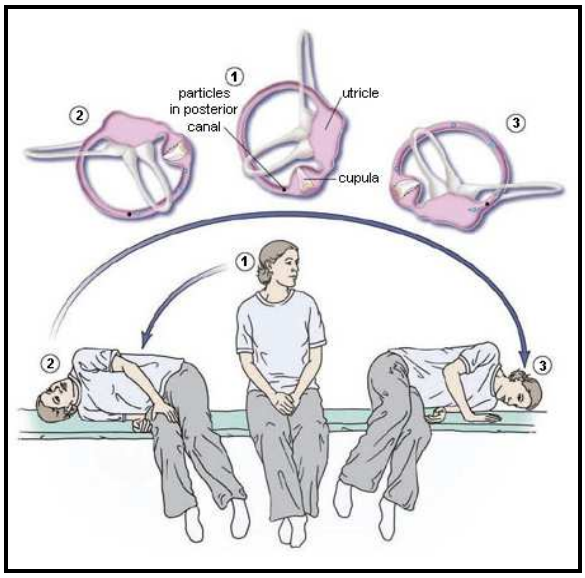

Figure 2: Liberatory Manoeuvre of Semont (Right Ear). Effect of the Manoeuvre on the Labyrinth as Viewed from the Front and the Induced Movement of the Canaliths (from Blue to Black). This Manoeuvre Relies on Inertia, so that the Transition from Position 2 to 3 Must be Made Very Quickly

Position; then the patient's head is turned 45 degree towards the unaffected side, and then is quickly put into a position lying on his or her side, toward the affected side, with his or her head turned upward nystagmus and vertigo may be observed. After about 5 minutes, the patient is rapidly moved to the opposite side-lying position without pausing in the sitting position and without changing the head position relative to shoulder. The patient remains in this second position for 5-10 minutes and gradually brought back to the upright sitting position. Semont and Colleagues found an 84% response rate after 1 procedure and a 93% response rate after a second procedure 1 week later[20]. Other studies have had response rates of 52%–90%[21-23] with recurrence rates of up to 29%. There has been no difference in efficacy shown between the liberatory manoeuvre and particle repositioning manoeuvre, as randomized studies by Herdman and colleagues[22] and Cohen and Jerabek[24].

Particle Repositioning Manoeuvre

This is the most frequently performed repositioning manoeuvre of the vertical canal that Epley published his first report on the "**canalith repositioning procedure**" (CRP).[25] During seated position mechanical skull vibration is routinely used and the patient's head is moved sequentially through 5 different positions. In this procedure the otolithic debris moves under the influence of gravity from the posterior semicircular canal into the utricle. Today clinicians thought to use a modified version of the CRP. One modified CRP is the particle repositioning manoeuvre (PRM) which is a 3-position manoeuvre that eliminates the need for sedation and mastoid vibration[26, 27] (Figure 3).

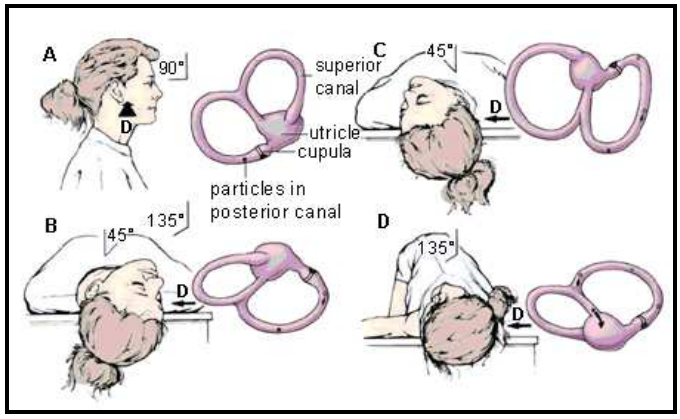

Figure 3: Particle Repositioning Manoeuvre (Right Ear)

In this procedure first patient is seated on a table as seen in the figure. Then patient in normal Dix-Hallpike head hanging position (B) and maintain this position for 102 minutes. Then patient head is rotated in opposite side with neck extended through position C and into position D. The patient's eyes are immediately observed for nystagmus. Position D is maintain for 1-2 minutes and then sits back to position A. Overall the PRM should take less than 5 minutes to complete.

Physiotherapist could be eligible for successfully carry out the PRM in most straightforward cases after good understanding of ear anatomy and patho-physiology of BPPV.

Effectiveness of CRP has been proved by Systematic reviews and Meta-analyses of Randomized Controlled Trials CRP have a very high level of evidence of effectiveness. Trial quality has been rigorously scrutinized by the Cochrane Collaboration,[28] the American Academy of Neurology Quality Standards Subcommittee,[29] a multidisciplinary guideline development panel,[30] and other independent groups.[31, 32]The summary results of all RCT indicates that CRP has a large effect size in treating patients with BPPV. In these studies, 61-80% of patients treated with CRP had resolution of BPPV compared with only 10-20% of patients in the control groups.[29]

HORIZONTAL (LATERAL) CANAL BPPV (HC-BPPV)

Diagnosis

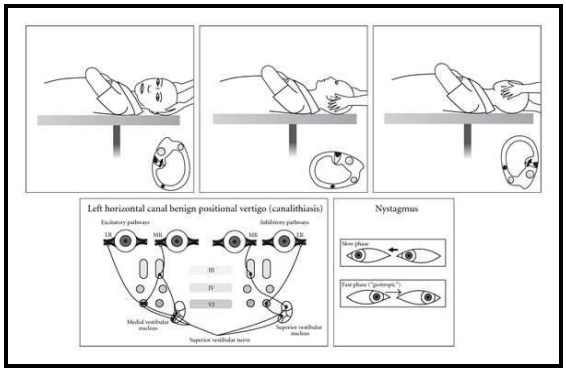

Figure 4: Horizontal Canal BPPV (canalithiasis) in a Left Ear Showing Head Roll Test, Inner ear, and Receptor Connections to the Extraocular Muscles

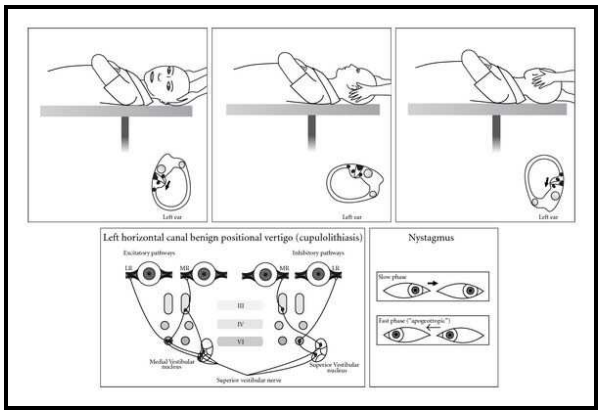

Figure 5: Horizontal Canal BPPV (Cupulolithiasis) in a Left Ear Showing Head Roll Test, Inner ear, and Receptor Connections to the Extraocular Muscles

The HC-BPPV has sudden onset and usually more intense symptoms that the posterior canal variant, persisting longer than 30 seconds, and is often associated with nausea and eventually vomiting. The diagnosis of HC-BPPV can be more challenging than posterior canal BPPV because it may be difficult to determine the affected side, and it is diagnosed by **Pagnini-McClure manoeuvre**, in which the patient's head is turned by one side while supine. During this maneuver, if horizontal nystagmus may beat toward the direction of head turn (referred to as geotropic nystagmus) (Figure 4) or in the direction opposite the head turn (called apogeotropic nystagmus) (Figure 5). Diagnosis of the affected side (lateralization) is very important for the planning the future treatment of HC-BPPV. Since ampullopetal flow of the endolymph evokes a greater response than ampullofugal flow in the horizontal canal (Ewald's second law), and the induced nystagmus mainly in the supine position which is stronger when the head is turned toward the affected ear in the geotropic type of HC-BPPV, whereas, stronger nystagmus is induced when head is turning towards the healthy ear in apogeotropic HC-BPPV. Caloric test can show hypoexcitability in the affected ear.

In HC-BPPV, nystagmus may be induced by **Bow and Lean test**, when the patient bows the head over 900 (bowing nystagmus) and leans the head backward over 450 (leaning nystagmus) in the sitting position. In up to 80% of HC-BPPV cases, bowing and leaning nystagmus are in the opposite direction. In geotropic HC-BPPV, bowing nystagmus beats mostly toward the affected ear (ampullopetal migration of the otoliths), while leaning is directed mostly toward the healthy ear

(ampullofugal displacement of the otoliths). In contrast, bowing nystagmus is mostly contralesional and leaning nystagmus is usually ipsilesional when observed in apogeotropic HC- BPPV. Bowing and leaning nystagmus in apogeotropic HC-BPPV are explained by deflection of the heavy cupula in response to the positional change.[33-36]

In apogeotropic HC-BPPV, the induced horizontal nystagmus may disappear when the head is turned to the affected ear by 10-20 degree, while supine (**null point**), [37] which is explained by alignment of the heavy cupula in the direction of the gravitational vector.

TREATMENT

Geotropic HC-BPPV

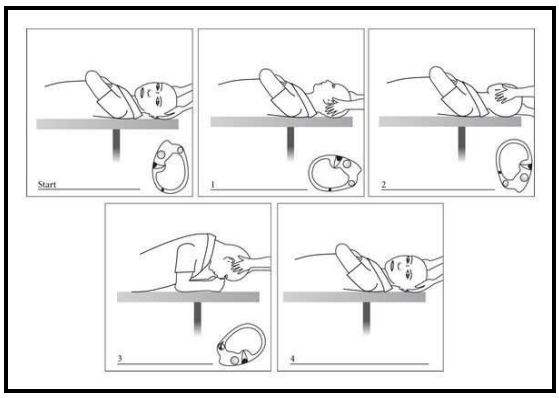

Figure 6: "Barbecue" Repositioning for Horizontal Canal BPPV in a Left Ear

Barbecue 360 degree Maneuver (Supine roll maneuver) for treatment of geotropic HC-BPPV. Patient's head is rolled 360 degree in quick 90 degree increments (with one minute intervals). This motion is started with a head rotation from the supine position to the unaffected side. Subsequently, the patient is rolled over to the prone position while the head is held in the same position before it turned rapidly to the nose-to-ground position. Then the head rotated vigorously to the opposite lateral position with the affected ear once again pointed towards the ceiling. Finally, the patient is sat upright with the chin tucked and the head is extended (Figure 6). During these maneuvers, the otoconial debris migrates in the ampullofugal direction, and entering the utricle through the non-ampullated end of the horizontal canal. Lying position in the healthy ear downward for approximately 12 hours (**forced prolonged position**) can be maintained, especially in a patients with severe symptoms and unable to perform sequential position changes.[38, 42]

The **Gufoni maneuver (there are a couple variation on the Gufoni maneuver)** is another alternative.[39, 40] After being seated on side of bed and then quickly lie down on the healthy lateral side and is stay in that position for 1-2 minutes until resolution of the evoked nystagmus. Then after patient quickly turn the head down into the bed with the patient maintaining this position for another 2 minutes, followed by a slow return back to the starting position.

Apogeotropic HC-BPV

In Apogeotropic HC-BPPV the induced horizontal nystagmus may disappears when the head is turned to the affected ear by 10-20 degree, while supine. The therapeutic goal should be to shift the debris from the anterior into the posterior arm of the horizontal canal[41]. If the otolithic debris is attached at the utricular side of cupula, its detachment should result in immediate resolution of the positional vertigo and nystagmus. A modified Semont maneuver, and the Gufoni method addition with head shaking in the horizontal plane have been proposed for the treatment regimens for apogeotropic HC-BPPV[42]. The modified Semont maneuver includes: 1) the patient is brought briskly into a side-lying position with the affected ear downward; 2) the patient's head is turned 45 degree downward, with this position being maintained for 2-3 min; and 3) the patient resumes the original sitting position. During Semont maneuver head shaking helps in to detach the otolithic debris from the capula.

In the Gufoni maneuver, the patient sits with the head directed straight ahead and then quickly moves into a side-lying position on the affected side, remaining in this position for 1 or 2 more minutes after the end of apogeotropic nystagmus. The head is then turned 45degree upward very quickly and is kept in this position for 2 minutes, followed by a

slow return to the sitting position. This Gufoni maneuver helps in to remove the otolithic debris from the anterior arm of the horizontal semicircular canal near the cupula.

ANTERIOR CANAL BPPV (AC-BPPV)

DIAGNOSIS

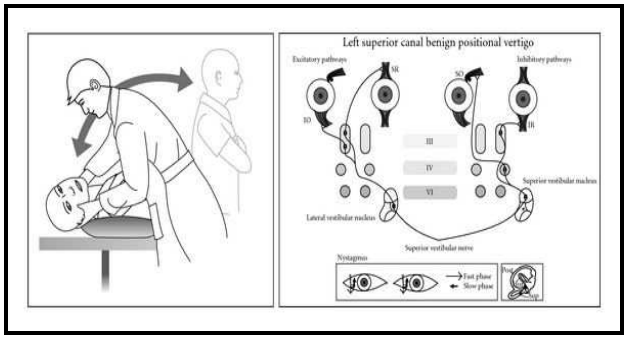

Figure 7: Superior Canal BPPV in a Left Ear Showing Dix Hallpike test, Inner Ear, and Receptor Connections to the Extraocular Muscles

Anterior-canal BPPV is considered the rarest form of semicircular canalolithiasis, with a postulated frequency of 1-2%. Its low incidence contrasts with the clinical importance of its most prominent characteristic, positional down-beating nystagmus, which also occurs as central positional nystagmus associated with various brainstem and cerebellar lesions, and may indicate a sinister pathology[43-44]. This down-beating nystagmus with an ipsitorsional component indicates the affected side. (Figure7). However the torsional component may

Not be evident by visual inspection alone and sophisticated three dimensional sclera-oil or video-oculographic recording are necessary.

TREATMENTS

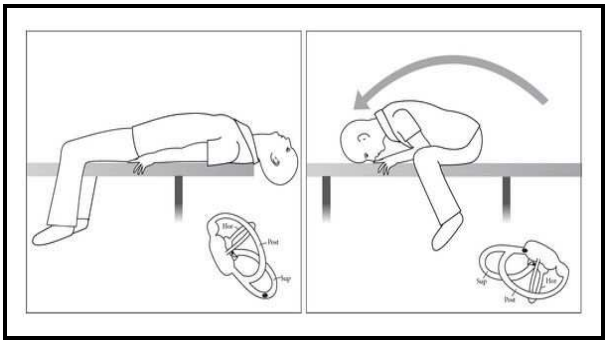

Figure 8: The Li Manoeuvre for Superior Canal BPV in Either Ear (Left Ear)

Grouping treatment maneuvers into requiring knowledge of affected side and those that do not yielded analogously high clearance rate. Posterior canalolithiasis can resolve symptom in 75.9%-95% of cases with the exception of Blakley study. The Epley and Semont maneuver success rates are similar with no study thus far showing a significant difference between the two. Modified repositioning maneuvers and forced prolonged position have also been adopted in treating this particular BPPV[45, 46]. Li maneuver [47] where the patient is moved rapidly from a supine (midline) head-hanging position to a face-down position at the opposite end of the couch (Figure 8).

REHABILITATION

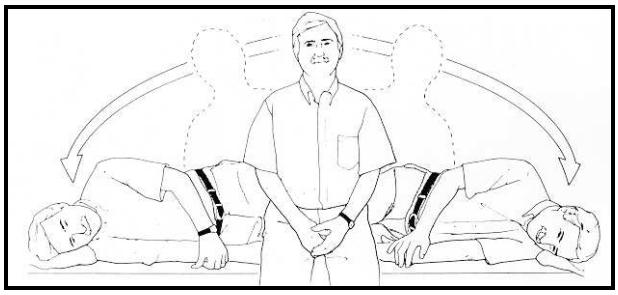

Figure 9: Brandt-Daroff Exercise. Patients are Instructed to Rapidly lie on their Side, Sit up, Lie on the Opposite Side, and then Again sit up. Each Position should be Maintained for at Least 30 Seconds. These Exercises are Repeated Serially 5-10 Times a Day Until Resolution of the Symptoms

Irrespective of the involved canals, the **Brandt-Daroff** exercise can be given instead of or in addition to the head movements carried out by the healthcare professionals (Figure 9). These exercise do not provide an instant cure for dizziness, instead a more gradual improvement would be seen as the exercises are repeated for 2-3 times per days and continue until patients have experience two consecutive vertigo-free days. In PC-BPPV, vestibular rehabilitation shows most effective treatment outcomes compared with placebo[48] and there are lack of evidence and data concerning the effectiveness of vestibular rehabilitation in case of HCBPPV.

ASSISTIVE DEVICES

Oscillators

A review of literature suggests that most researchers and clinicians have not found the vibrator to be critical component in the treatment of BPPV. There are also other options have reported the use of oscillation over the mastoid bone during the treatment procedure to facilitate movement of debris. One study[49] reported that mastoid oscillation was critical for success, however, only a single maneuver was performed prior to determining the outcome. Other studies[50] demonstrate excellent success rate treatments for PSC BPPV without the use of oscillation, and in a direct comparison no additional benefit was found[51].

CONCLUSIONS

Good balance is essential for daily life, from getting out of bed to crossing the road. Balance disorders and dizziness are a growing public health concern across all age groups. BPPV is a common problem, and will be encountered more and more as our population ages. The impact can range from a mild annoyance to a highly debilitating condition, and can affect function, safety, and fall risk. With the help of trained healthcare professional diagnosis and intervention of BPPV, can be easily possible and world can stop spinning.

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